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Genetic Effect of Small Doses of Ionising Radiation

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The estimate of the genetic effect of small doses of radiation on human heredity is a very complicated problem. Its solution requires preliminary model experiments on various objects as well as irradiation experiments on human cells in tissue cultures.

The cumulation of genetic effects of fractioned and chronic irradiation with small doses

The genetic effect of small doses of gamma-rays (Co-60), fast neutrons with the energy of 1 Mev and protons of 126-130 Mev has been studied on the pattern of lethal and sublethal sex-linked mutations in Drosophila melanogaster.

These experiments had to solve the following problems:

a) the comparative mutagenic effect of γ -rays, fast neutrons with the energy of 1 Mev and protons of 126-130 Mev at the level of small doses of radiation;

b) the comparative effect of a single and fractioned irradiation with small doses;

c) the dependency of the frequency of mutations induced by small doses from the stage of spermatogenesis;

d) whethem 5 rads and 2 rads are threshold doses for genetic effect;

e) is there the cumulation of mutagenic effects at such low doses and big intervals between irradiation treatments as well as at the chronic irradiation.

Principal experiments were aimed at discovering the mutagenic effect of a single irradiation with the intensity of 20 rads and fractioned irradiation of the same intensity in four irradiation treatments with the dose of 5 rads in each treatment with intervals of an hour and a half. In experiments with fast neutrons, in addition to it, the mutagenic effect of fractioned irradiation was studied as well, the dose of each of four treatments being 2 rads, the whole dose of

irradiation - 5 rads and the intervals between them being one hour and a half.

γ -raying from Co-60 was performed in a specially designed irradiation installation with the fixed distance between the Co-60 preparation and the irradiated point in gelatinous ampules with flies (24 cm). At the beginning of the operation the intensity of the irradiation was equal to 0.93 rad per minute, the duration of the irradiation with the dose of 5 rads being 5 minutes 24 seconds. With the decay of Co-60 and the decrease of its intensity the duration of treatments was being correspondingly amended.

The irradiation with fast neutrons at a level of 1 Mev was performed in one of the reactors of the Academy of Sciences of the USSR with the efficiency of 1000 kV. Flies were irradiated at the moment of the emergence of the beam from the beam tube. The dose rate in the first experiments was 115 rads per hour, thus, the treatment with a dose of 5 rads required 2 minutes 36 seconds. In later experiments the dose rate was $\sim$60 rads per hour which made it possible to achieve high accuracy in treating with even such small doses as 2 rads.

The irradiation with protons of 126-130 Mev was performed in a synchrocyclotron of the United Institute of Nuclear Research in Dubna. The intensity was $\sim$ 0.3 rad per second, the 5 rad treatment lasting 17 seconds.

The genetic effect of the small dose irradiation was expressed as a sum of induced lethal and sublethal mutations. The whole number amounted to 341 letnals and 30 sublethals in the experiments and 42 lethals and 1 sublethal in the control. All mutations were checked by corresponding control crosses.

The experiments were carried out on the wild strain of the D-32 type which had been specially bred to obtain the low spontaneous mutability in sex-linked recessive lethals.

During 1959-1962 the occurrence of such spontaneous mutations was: in the progeny of males 1-3 days old - $0.050 \pm 0.02\%$, in the progeny of males 7-8 days old - $0.079 \pm 0.028\%$. In 1963 a certain increase of the sponta-

neous mutability of the D-32 strain was observed: in the progeny of males 1-3 days old - 0.13 ± 0.035%, in the progeny of males 7-8 days old - 0.11 ± 0.039%, in the progeny of males 17-20 days old - 0.06 ± 0.023%. The corresponding data of spontaneous mutation occurrence were substracted in the calculations for all experimental treatments.

The data on the effect of radiation are shown in Table 1. It is seen that the dose of 5 rads is not a threshold dose for γ -rays, fast neutrons of 1 Mev and protons of 126-130 Mev because the fractioned irradiation consisting of four 5 rad treatments resulted in the cumulative mutagenic effect similar to one which is usually obtained after the corresponding treatment at a level of 20 rads. Thus, a single γ -raying at a level of 20 rads induced 0.105 ± 0.040% sex-linked recessive lethal and sublethal mutations in spermia, while four successive treatments each at a level of 5 rads induced 0.162 ± 0.051 % mutations. The corresponding data for fast neutrons were 0.259 ± 0.060% and 0.242 ± 0.058% . The same phenomena are observed for these two types of radiations with regard to their mutagenic effect on spermatids.

The data in Table 1 show that even the dose of 2 rads is not a threshold dose for fast neutrons. For example, four successive treatments of spermatids at such doses with intervals of one hour and a half resulted in a greater number of induced mutations. A number of mutations after the treatment at a total level of 8 rads approximately conforms to 40% of a number of mutations expected at a level of 20 rads.

The similar treatment of spermia and spermatogonia showed some increase of a number of mutations but the data obtained are not statistically significant. The proton treatment of spermia resulted in increase of a number of mutations , which may be regarded as a statistically significant one.

The mutagenic effect of small doses of fast neutrons (per one rad) is 1.5 - 3 times higher than that of γ -rays, the mutagenic effect of 126-130 Mev protions is somewhat lower (0.5 - 0.8) than that of γ -rays. The relative mutagenic effect of small doses of these three ionising sources is

equivalent to the relative mutagenic effect of high doses of these types of radiation. For example, the mutagenic effect (estimated on sex-linked recessive lethals) of 1 Mev fast neutrons at a level of 1000 rads is 1.5 times higher than that of γ -rays, and the mutagenic effect of 126-130 Mev protons is 0.4 - 0.8 of the mutagenic effect of γ -rays (1,2).

The comparison of a number of sex-linked recessive lethals and sublethals per 1 rad/10^7 gametes induced by the irradiation with small doses and by a single irradiation at a level of 1000 rads (Fig.1) shows that small doses (20 rads in one treatment or fractioned irradiation, 5 rads in each treatment) produce far more point mutations, which are mainly recessive lethals, than the high doses. This fact proves that the genetic danger of small doses of irradiation is much greater than it could be expected from extrapolating data obtained on high doses.

E.Guyenot and oth. (3) performed a fractioned irradiation of Drosophila males with X-rays at the dose of 20 rads; they induced per 1 rad/10^7 gametes 456 sex-linked recessive lethals; T.Shiomi and oth.(4) performed a single irradiation of Drosophila males with X-rays at the dose of 8 rads and induced per 1 rad/10^7 gametes 537 similar mutations. These data correlate with the corresponding data on small doses of γ-rays obtained in our experiments: 525 in a single treatment and 810 in a fractioned treatment at the dose of 20 rads.

In our experiments a single treatment of spermatogonia with γ -rays at a level of 1000 rads produced only 20 sex-linked recessive lethals and sublethals induced per 1 rad/10^7 gametes while a single treatment of spermatogonia with 1 Mev fast neutrons at a level of 20 rads and a fractioned treatment at a level of 8 rads produced 420 and 75-260 of them respectively. The irradiation effect of spermatogonia is the most durable , so the increased sensitivity of this stage of spermogenesis to the mutagenic effect of fast neutrons makes the latter particularly dangerous.

The chronic irradiation with γ -rays was performed in two variations. In the first variation, eggs, larvae and pu-

pae were treated. In the second variation, flying males were treated. In both variations of this experiment the chronic irradiation lasted 11-13 days, the γ-ray dose in different series was 8.3 - 10.0 rads daily, the average cumulative dose of the chronic treatment being 108 rads for the period of development and 113 rads for imago males. The temperature of all γ-ray treatments was 20-22°C.

During these experiments the rate of sex-linked recessive lethal mutations induced by the chronic irradiation during the development period was equal 0.224 ± 0.05 per cent, i.e. 207 lethals occurred for 1 rad/10^7 gametes. During the irradiation on the imago stage 0.183 ± 0.07 per cent of lethals were induced, i.e. 102 lethals for 1 rad/10^7 gametes. These data correlate with those cited by D.E.Uphoff and C.Stern (5) and W.P.Spencer and C.Stern (6), where a number of sex-linked recessive lethals induced by the chronic irradiation of Drosophila males at the doses of 50-100 rads per 1 rad/10^7 gametes was equal to 155-293. It is evident that there is the cumulation of mutations induced by small doses.

The comparative cytogenetic radiosensitivity of monkey and mice

Radiogenetic and radiocytologic studies on different mammal species are of great importance both per se and for an estimate of the effect of radiation on human heredity.

Many scientists successfully used mice for such studies. An important step was made with the discovery of the fact that hereditary structures in cells of monkeys are more sensitive to the radiation effect than those of mice.

New experiments, which are described below, were undertaken to study the cytogenetic effect of radiation at a level of 10-25 rads (the dose rate of 10 r/m) on the spermatocytes I. The quantity and types of chromosome rearrangements were analyzed by metaphases and anaphases in meiotic divisions. The monkey' gonads were irradiated locally, mice received the whole-body irradiation. Monkeys were castrated and mice were killed on dates which corresponded with the duration of meio-

tic stages in monkey (7) and mice (8). Three later meiotic stages were studied: late pachynema, diplonema and diakinesis + metaphase I. The frequency of chromosome rearrangements in control monkeys was 0.74 % and in control mice - 0.83 %. After the irradiation the frequency of chromosome rearrangements in the first meiotic division in monkey was higher than that in mice. The stage of diakinesis + metaphase I proved the most sensitive stage of the meiotic prophase to the effect of ionising radiation.

Fig.2 shows the data obtained. It is established that the higher cytogenetic radiosensitivity of monkeys as compared with mice retains its validity at a level of 25 and 10 r as well. It is interesting to note that there is a great relative effect of the dose of 10 r both for monkey and mice at all investigated stages of the prophase of meiosis. For example, at the stage of diakinesis + metaphase I a number of induced chromosome rearrangements per 1 r in monkeys is: at the dose of 25 r - 0.215 %, at the dose of 10 r - 0.328 %.

When we add these new data on the cytogenetic effect of X-rays at the dose of 10 and 25 r with earlier data on the effect of 50 r and more (9, 10, 11, 12) we obtain the degree of the cytogenetic radiosensitivity of different phases of the prophase of meiosis in monkeys and mice summed up in Table IIa.

In all above-mentioned experiments the asymmetric rearrangements of chromosomes were studied. These rearrangements do not pass the II meiotic division and are not transmitted to spermatids and spermia. It is of great interest to observe symmetric translocations which pass the meiosis and are found in gametes. These rearrangements can be recognized by their configuration and by the quantity of bivalents in the metaphase of meiosis (13). Mice have 20 bivalents at the normal complex (one of their bivalents consists of a chromosome pair X-Y), monkeys have 21 bivalents (among them a pair X-Y). In this experiment animals were irradiated with the dose of 50 r, monkeys being castrated 24 hours later, mice - 21 hours. The number of symmetric translocations is shown in Table IIb.

The quantity of chromosome rearrangements, which were transmitted to the progeny and which are known to cause the

partial sterility in it, the destruction of linkage groups, the position effect and other changes also showed that the radiogenetic sensitivity in monkeys is 2 times higher than that in mice.

The fact that the difference in cytogenetic radiosensitivity of monkey and mice, proved by our experiments, is observed at small doses of radiation as well is of great importance. It is evident that the data on the effect of small doses of radiation on the heredity of man can be extrapolated from other mammal species only with great caution. The comparison with the experimental data on man's cells in tissue culture shows that the genetic radiosensitivity of monkeys is nearer to man as compared with mice. For example, the doubling dose for diakinesis in mice should be 8 rads, for diplonema - 10 rads, for pachynema - 17 rads. Meanwhile, the corresponding dosage for monkey is: 3 rads, 4 rads and 6 rads.

The effect of small doses of radiation on human cells in tissue culture

The problem of an estimate of the effect of small doses of radiation on cells cannot be solved without the knowledge of the total time and duration of separate phases of the cell cycle because different phases of the cell cycle are characterized by different sensitivity. The normal cycle of man's euploid cells in culture lasts 24 hours, the duration of phases in hours being as follows: G_1 - 9; S - 8; G_2 - 6; M - 1 (14). It is known that X-raying at a level of 25 and 50 r extends the duration of the cell cycle to 70 hours (15, 16, 17).

In order to study the cytogenetic effect of the doses of 5, 10, 15 and 20 r it is necessary to know their effect on the cell cycle. Presently there are no published data of this kind. In the course of analysis of the mitotic index by counting colchicinized metaphases after the irradiation of human cells in the initial culture with different doses of gamma-rays the dependence was showed between the duration of the cell cycle and the level of doses. Fig.3 shows that the gamma-ray doses of 5-20 r practically do not influence the mitotic activity of human cells in culture and consequently do not change

the duration of the cell cycle. The treatment with the doses of 25-50 r suppresses the mitotic activity of human cells in tissue cultures, extending the cell cycle to 60 hours.

In order to analyze the chromosome rearrangements induced by the doses of 5-20 r the man's cell cultures were fixed 2, 6, 10, 14 , 18, 30 and 45 hours after the irradiation. Under the conditions of the normal cycle the fixation after 2 and 6 hours gave us data on the quantity of rearrangements induced at the irradiated presynthetic phase (G_1); the fixation after 10 and 14 hours - at the phase S (the synthetic phase); the fixation after 18 hours - at the phase G_2 (the post-synthetic phase). The fixation after 30 and 45 hours coincides with the second mitosis after the irradiation. At anphases we analyze only asymmetric rearrangements which die during mitosis. Therefore such radiation induced rearrangements are not expected to be observed in the second mitosis.

The analysis of chromosome rearrangements showed that all these suggestions were right. The fixation 2 and 6 hours after the treatment resulted only in chromatid rearrangements. Chromosome translocations were observed after 10 hours. The fixation after 30 hours resulted in the quantity of chromosome rearrangements which did not exceed that in the control.

The treatment with all above-mentioned doses, beginning from the dose of 5 r, resulted in the significant increase of the quantity of both chromatid and chromosome rearrangements. The fixation 5 hours after the treatment gave the following picture of radiation induced rearrangements (per cent per 1 rad): the dose of 5 r - 0.52 ; 10 r - 0.11; 15 r - 0.13 ; 20 r - 0.10 . The average percentage for the stage G_2 is 0.21 % per 1 rad. The fixation after 14 hours gave: the dose of 5 r - 0.11; 10 r - 0.26; 15 r - 0.07; 20 r - 0.09. The average percentage for the stage S is 0.13 % of rearrangements per 1 rad. The fixation 14 hours after the treatment which corresponds to the stage G_1, resulted in the percentage of rearrangements per 1 rad averaging 0.25 %. The medium for all treatments amounts to 0.20 % of rearrangements per 1 rad. The quantity of chromosome rearrangements in the

control averages 1.50 %. Thus, the dose of radiation which doubles the number of mutations in human cells in tissue cultures as compared with the natural mutation process is equal to 7.5 rads.

Such level of the doubling dose obtained for the first time as a result of the analysis of principal phases of the cell cycle at small doses of radiation coincides on the whole with the data obtained by N.P.Dubinin (18) and N.P.Dubinin, Yu.Ya.Kerkis and L.I.Lebedeva (19), when the doubling dose was showed as equal to 10 r. It is proved now that the level of the doubling dose is even lower. The most prolonged pre-synthetic phase (G_1) requires 6 rads.

The problem of protection against small doses of ionizing radiation

There are numerous publications (see 20) on the problem of chemical protection against the damaging effect of ionizing radiation both by lowering the lethal dose for an organism and by partial removal of the effect of radiation on hereditary structures. However, the problem of the efficiency of protection with regard to dosage has not been studied at the cytological level. According to the widely accepted opinion, the increase of the dose is accompanied with the monotonous decrease of the efficiency of protection at the cellular level (see 21).

We investigated the protective efficiency of AET (β -amino-ethilisothiouronium), serotonin and streptomycin introduced into human cells in tissue culture prior to their irradiation with γ -rays with dose of 25, 50 and 100 r. The cytogenetic effect of gamma-rays on human cells in tissue cultures is about 50 per cent lower as compared with that of X-rays (17). Thus the irradiation with γ -rays at a level of 25 r produces the same genetic effect as that with X-rays at a level of 12.5 r.

There were 106206 anaphases studied in the experiment. Such scale of the experiment provided significant results. The latter included all types of chromatid and chromosome

rearrangements in all principal phases of the cell cycle (G_1; S; G_2) and separate parts of the cycle within the phases. As the duration of the cell cycle with the cells are irradiated with 25, 50 and 100 r, is 60-70 hours, the cultures were fixed after the treatment for 11 periods from 2 to 70 hours. The radiosensitivity curves, being similar in some features, differed, depending on different doses of irradiation, in the quantity of chromosome rearrangements and in the rate of the replacement of chromatid rearrangements by the chromosome ones. Similar curves were obtained - when the protective substances were used 20-30 minutes before the irradiation with the doses of 25, 50 and 100 r. The experiments showed that, when the protective substances were used, there were different responses of chromosomes to the irradiation with different doses of γ -rays.

Protective substances applied before 50 r treatment produced an evident picture of protection. The degree of this protection depended on phases of the cell cycle. Average data for the whole cycle in the 50 r treatment variation were: for serotonin - 43 per cent of protection; for streptomycin - 43 per cent; for AET - 37 per cent. The maximum protection was observed at the phase of synthesis (S). The corresponding data for this phase were: 60;57 and 45 per cent. In the 100 r treatment some protective effect was also produced, but the degree of that was not the same. The efficiency of streptomycin was evidently lower, it did not exceed 25 per cent; the efficiency of serotonin was almost the same - 40 per cent of protection; the efficiency of AET proved higher - 50 per cent of protection.

The irradiation with γ -rays at the dose of 25 r resulted in absolutely different data. Serotonin showed zero efficiency for the whole cell cycle; the same was the result of the pre-treatment application of streptomycin and AET.

Thus protective chemicals can modify the effect of the irradiation of hereditary structures in human cells with certain doses. Protective compounds produce different effect on the irradiation with different doses. At the same time there is an unexpected fact that the protective substances applied provided no protection to the hereditary structures when cells were treated with small doses of radiation.

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These data require thorough studies in those directions. It is necessary to try more groups of protective chemicals; to extend the knowledge of the relation between protective effects and the dose of irradiation at the choromonomal level; to reveal the forms of this relation not only for chromosome rearrangements but for gene mutations as well. The corroboration of the non-efficiency of available forms of chemical protection of hereditary structures against the effect of small radiation doses would mean that radiobiology is going to face a new important task. It is necessary, developing our knowledge of initial mechanisms of the radiation effect on mutagenesis, to find the new means of the protection of heredity against the chronic influence of small doses of radiation.

The genetic effect of the radionuclide Sr-90

It is known that strontium-90, cesium-137 and carbon-14 are the most dangerous for living organisms of all radioactive isotopes left after atomic and thermonuclear explosions. The mutagenic effect of Cs-137 and C-14 is out of doubt. The genetic effect of Sr-90 is a much more complicated problem. The determination of the genetic effect of Sr-90 on any object of studies is of principal importance . In order to approach the problem Sr-90 was studied with regard to its effect on the unicellate green algae Chlorella. There were four strains used in the study bred at the Laboratory of radiogenetics of the Institute of biophysics of the Academy of Sciences of the USSR; two of them - LARG-1 and 138 belong to species Chl.vulgaris; the strain LARG-2 belongs to species Chl. ellipsoideal and the strain 36 - to species Chl. terricola species. LARG-1 is the most big-celled strain of the four. The dry weight of 100.10^6 cells of this strain is 3.20 mg; it is followed by the strain 138-0.70 mg; LARG-2 - 0.56 mg and the strain 36-0.40 mg.

The material from initial trays was seeded into 250 ml conic flasks, containing 100-150 ml of the Tamiaja medium; after the preliminary growing at the light intensity of about 8000 luxes for 3-5 days (t^o = 30-34°C) the culture was synchronized and prepared for treatments (22). The compaction of the suspension of Chlorella cells was brought to 10 mill/ml

after which 1 ml of the suspension was treated with 1 ml of the active solution of Strontium nitrate-90. The dose of β-radiation was defined from the activity of strontium-90 in 1 ml of the treated suspension with the compaction of 5 mill/ml of cells. The cultures were kept in darkness for 24 hours, then the cells were washed to free them of strontium-90 (externally) and seeded into the agarized medium. The survival data were determined microscopically (x 200 - x 400) by counting those cellular regions which were able to develop viable clones on the agar. Mutations were counted after seeding cells on Petri dishes, 500-1000 colonies on each dish. The seeded cultures were grown in luminostates under the above-mentioned conditions.

The survival data for all strains are shown in Fig.4 . It is seen that all strains have clearly expressed sigmoid 'dose-effect' curves. The Glocker's index for LARG-1 and LARG-2 strains was about 20. Exponential survival curves which had been obtained earlier as a result of irradiation with X-rays of the same strains showed that Chlorella is a haploid organism.

The data on the mutability of the LARG-1 strain treated with strontium-90 are shown in Table III. The mutational nature of principal changes, shown in Table III, was studied in special experiments. The irradiation with X-rays resulted in curves of direct dependence of mutability rate on a dose. The strontium-90 treatment showed that the increase of a dose resulted in the increase of the mulability rate in proportion to some part of the dose.

It is found that coefficients of strontium-90 accumulation are growing higher with the increase of the activity of cell suspensions. For example, at the activity of 0.1 mccurie/ml the strain LARG-1 had the coefficient of accumulation equal to 514 , the strain LARG-2 - 3240; at the activity of 1.0 mccurie/ml the corresponding coefficients were 821 and 5070; at the activity of 10.0 mccurie - 1710 and 6200. So high accumulation coefficients lead to the high intracellular irradiation.

Thus, the Glocker indices of the 'dose-effect' curves and the increase of mutability in proportion to some part of the applied dose can be explained by the increase of accumulation coefficients.

It is shown (24) that the radionuclide Sr-90 is being included into chromosomes replacing Ca there. It is necessary to go on with wide studies of genetic effects of the inclusion of Sr-90 into the chromosomes of somatic and germ cells in animals and plants.

CONCLUSION

The experimental data of this work were obtained by the authors in experiments on the effect of different types of ionizing radiations on the hereditary structures in cells of monkeys, mice, Drosophila, on human chromosomes in tissue cultures as well as on the mutagenic effect of the radionuclide Sr-90 in experiments in unicellate green algae. These data give a quantative and qualitative estimate of the genetic effect of small doses of radiation.

The genetic effect of small doses of radiation is of a clear cumulative nature. The experiments on fractioned and chronic irradiations with the above-mentioned dose rates at the level of small doses did not show the phenomena of the reparation of genetic injuries. Moreover, there are some cases which indicate that small doses are relatively (per 1 rad) more efficient with regard to cytogenetic injuries. Presently it should be considered a proved fact that all principal long-life radionuclides left after nuclear explosions - Cs^{137}, Sr^{90} and C^{14} - are certain to produce genetic effects.

The dose of energy which doubles the spontaneous mutation process in man is 10-5 rads. It is found that the efficiency of chemical protection against the damaging cytogenetic action of radiation undoubtedly depends on the dose of radiation. It is proved that the tried protective chemicals have no protective ability against small doses of radiation.

The qualitative determination of the genetic effect of radiation suggests that any small dose produces a certain quantity of mutations in propotion to a level of a treatment, i.e. to the amount of energy which is absorbed by a cell.

It results in a certain change in human heredity which manifests itself in man's progeny. For mankind as a whole the burden of hereditary detects which result from the action of small doses of radiation on germ cells is a quite significant magnitude (see 25). Hence it is clear that any uncontrolled increase of the level of radiation in mankind's environment should be forbidden. First of all it concerns tests of atomic and nuclear weapon.

The atomic energy as a whole should serve peaceful purposes. Its use in genetics and breeding will make it possible to control the heredity of plants, animals, microorganisms and viruses.

As to the heredity of man there are only damaging genetic effects of radiation there. Hence our task is to take all measures against the increase of the background of radioactivity, to work out and to take protective measures against damaging genetic effects of all the kinds of ionizing radiations.

The discovery of the fact that all available forms of chemical protection are not probably efficient against small doses makes us to look for an absolutely new approach to the problem of the protection of man's heredity against the chronic effect of small radiation doses. This discovery makes the problem of the genetic effect of small doses of radiation on heredity especially urgent. It is necessary to turn attention of the uptodate radiobiology to the problem of the most accurate and full appraisal of genetic effects of small radiation doses on heredity.

It is necessary to undertake large-scale investigations in order to disclose the nature of primary mechanisms of radiomutagenesis at molecular and chromosomal levels. The latter is necessary in order to find new approaches to the problem of the efficient chemical protection of hereditary sturctures aggins the damaging effect of radiations.

Table 1

Occurrence of sex-linked recessive lethal and sublethal mutations induced in male gametes of Drosophila melanogaster D-32 strain irradiated with small doses of different types of ionizing radiations - without natural mutations

Type of radiation	Dose (rad)	Type of irradiation treatment	Spermia		Spermatids		Spermatogonia	
			Number of studied chromosomes	Induced mutations (% ± m)	Number of studied chromosomes	Induced mutations (% ± m)	Number of studied chromosomes	Induced mutations (% ± m)
γ-rays	20	Single	12889	0.105±0.040	13632	0.141±0.048	-	-
Co-60	20	Fractioned (5 rads x 4)	11306	0.162±0.051	13154	0.156±0.050	-	-
Fast	20	Single	9700	0.259±0.060	8673	0.221±0.061	9638	0.084±0.026
neutrons	20	Fractioned (5 rads x 4)	9581	0.242±0.058	13980	0.199±0.053	-	-
1 Mev	8	Fractioned (2 rads x 4)	24802	0.047±0.031	24014	0.116±0.037	13840	0.018±0.033
Protons 126-130 Mev	20	Fractioned (5 rads x 4)	8403	0.125±0.065	3540	0.084±0.085	1824	0.157±0.112

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Table IIa

Comparative radiogenetic effect of X-rays in spermatocytes I in monkeys and mice at the dose of 10, 25,50-400 r

		Diakinesis metaphase I	Diplonema	Late pachynema	Middle pachynema	Early pachynema
In average 1 r	Monkeys	0.26	0.17	0.11	0.11	0.14
	Mouse	0.10	0.08	0.05	0.04	0.05

Table IIb

Comparative radiosensitivity in monkeys and mice by the occurrence of symmetric translocations (dose - 50 r)

Kariotypes	Translocations - percentage	
	Monkeys	Mouse
Normal: 20 bivalents in mice, 21 bivalents in monkeys	64	81
One translocation (19 bivalents im mice, 20 bivalents in monreys)	22	13
Two translocations (18 bivalents in mice, 19 bivalents in monkeys)	4	1.3
Complex translocation and other types of changes	10	4.7
Changes - total	36	19

Table III

Mutations induced by Sr^{90} in Chlorella strain LARG-1

Dose mcurie/ml/day	Colonies investigated	Mutations observed				Total	Mutations %
		dwarf	pigment	spotted	morphological		
Control 0.0	5342	9	45	15	6	75	1.40±0.16
1.0	4118	8	38	8	3	54	1.31±0.17
3.0	5034	18	78	17	4	117	2.34±0.21
5.0	5553	26	56	29	6	117	2.10±0.19
7.0	3918	7	74	13	3	97	2.46±0.25
8.5	1923	12	39	10	3	64	3.30±0.41
10.0	7098	31	212	53	33	329	4.65±0.25
12.0	1254	9	42	10	5	67	5.27±0.63

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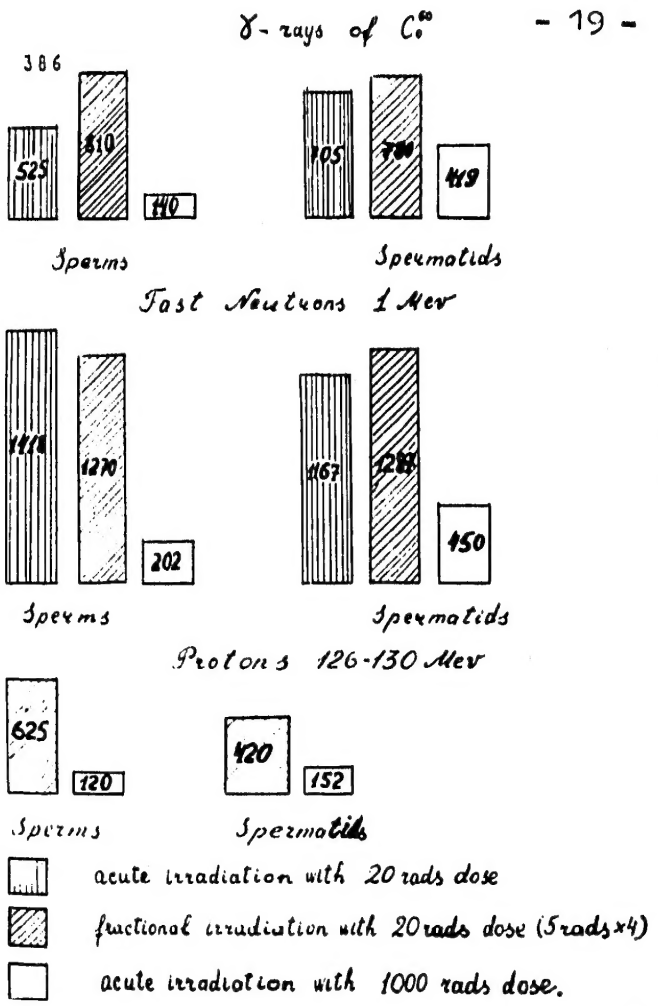
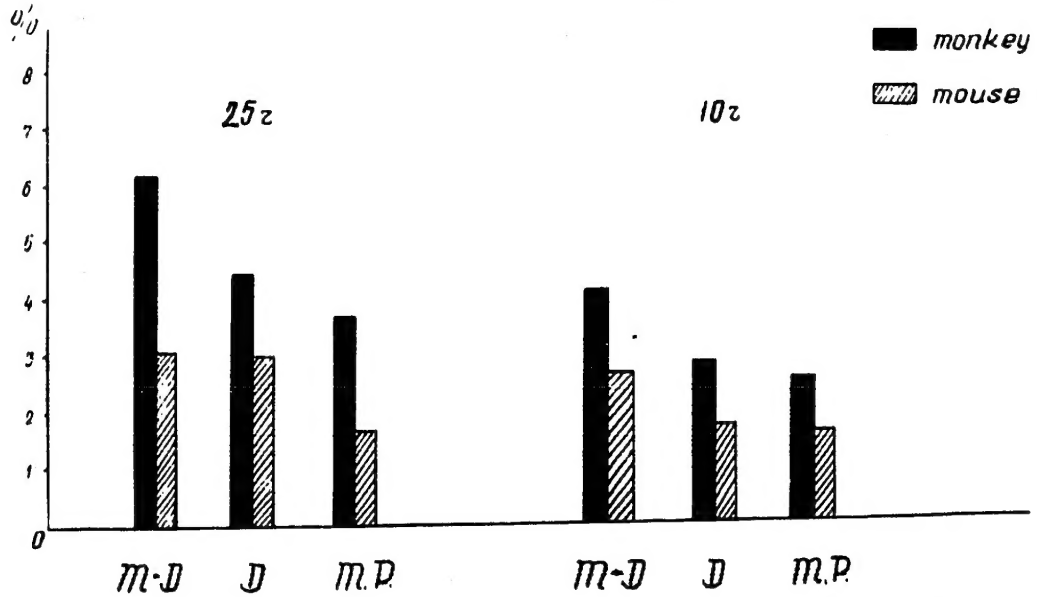


Fig.1

The quantity of sex-linked recessive lethal mutations induced by 1 rad per 10^7 gamete at small doses and at the dose of 1000 rads in one treatment



Percentage of chromosomal aberrations at first meiotic division after irradiation with 25 and 10 r of primary spermatocytes of mouse and monkey.

Fig. 2

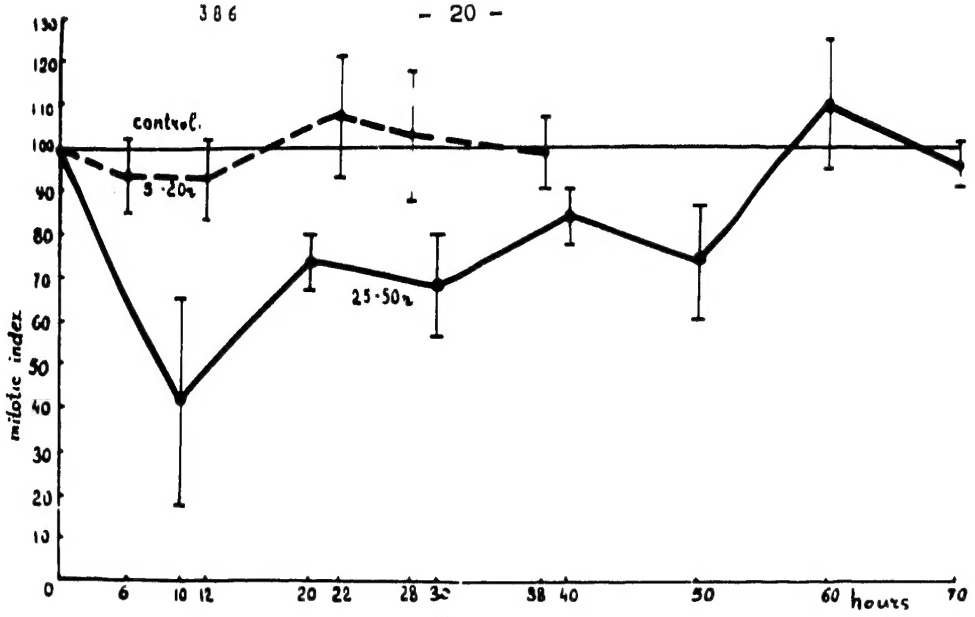


Fig. 3 Change of the mitotic activity in the culture of human cells after gamma-radiation at the dose of 5-20 r and 25-50 r

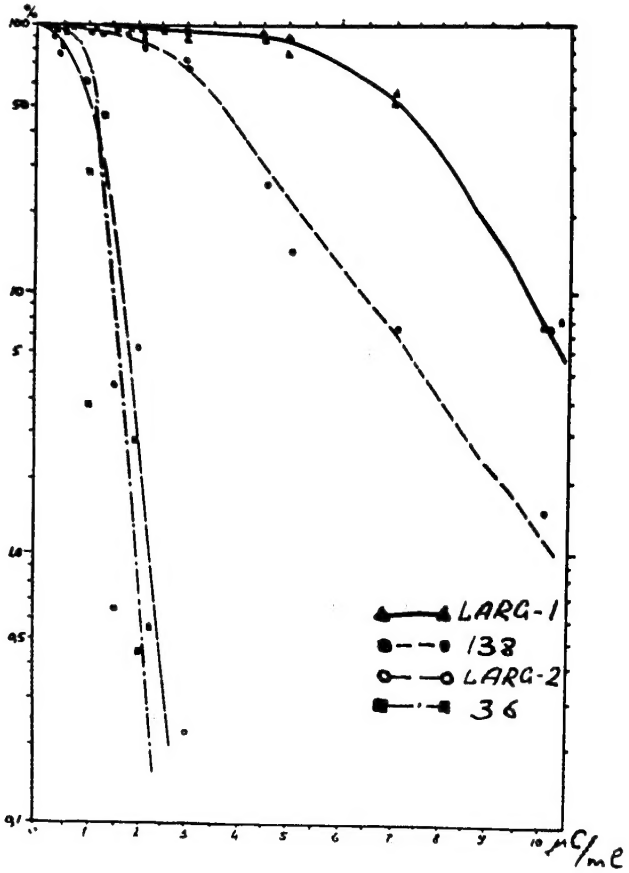


Fig. 4
The "dose-effect" curves for four Chlorella strains (LARG-1, LARG-2 and 36) treated with gamma-radiation of strontium-90